Cardiac Valve Replacement in Patients Over Sixty

A Report of Ten Operations with One Death

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■ *Of 85 patients who underwent cardiac valve replacement, 10 were 60 or more years old. Nine of the 10 lived, and the excellent results confirmed that advanced age does not contraindicate major corrective operations.*

MAJOR SURGICAL procedures are believed to be more dangerous in elderly patients since cardiopulmonary reserve tends to decrease progressively with age. It would be reasonable to assume, therefore, that open-heart operations in older patients would be associated with prohibitive mortality.[7] Our recent experience suggests, however, that persons in the seventh decade of life can tolerate major cardiac procedures with good results if appropriate precautions are taken.

Clinical Material

This report presents the results of operation in five male and five female patients 60 or more years old who were part of a total series of 85 patients with cardiac valve replacement. A cardiac valve was replaced in all ten patients and in two of the patients an additional corrective procedure was performed on another valve. Because of the limited life expectancy in this age group and the anticipated higher operative risk, every patient accepted for operation had Class III or IV cardiac dysfunction and was in danger of dying unless the condition of the heart could be improved by mechanical correction. Coronary artery disease, renal impairment, pulmonary disease or other factors which might increase the surgical risk were not regarded as contraindications to operation.

Methods

Perfusion. Total body perfusion was required for 61 to 182 minutes at flow rates ranging from 1.8 to 2.7 liters per square meter of body surface per minute, with moderate hypothermia (25°C). A disposable bubble bag oxygenator* primed with modified Ringer's solution was used. Deliberate hemodilution to a mixed hematocrit of 22

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*Manufactured by Travenol Laboratories, Morton Grove, Illinois.

TABLE 1.—*Clinical Data on Ten Cases*

Case No.	Age and Sex	Diagnosis	Cardiac Class	Significant Catheterization	Valve Replaced
1	65 F	Mitral stenosis; mitral insufficiency; tricuspid insufficiency; pulmonary emboli	III-IV	Cardiac index 2.6 Left atrium 33 — Pulmonary artery 68/32, 44	Mitral
2	61 M	Mitral stenosis; mitral insufficiency; tricuspid insufficiency; commissurotomy 1960	IV	Cardiac index 2.3 Left atrium 32 Pulmonary artery 80/35, 52	Mitral; tricuspid annuloplasty
3	60 M	Aortic stenosis; aortic insufficiency; mitral stenosis	III	Cardiac index 1.6 Left atrium 25	Aortic; mitral commissurotomy
4	62 M	Acute aortic insufficiency; bacterial endocarditis; mycotic embolus; obstructive airway disease	IV	Left atrium 23 Right ventricle 60/2	Aortic (Magovern)
5	63 M	Aortic stenosis; aortic insufficiency; myocardial infarct	III	Cardiac index 2.6 Left atrium 22 Pulmonary vascular resistance 2	Aortic
6	70 F	Luetic aortic insufficiency	III	None	Aortic
7	65 F	Severe aortic stenosis; angina; renal tuberculosis; nephrectomy	III angina	Aortic gradient 150 Left atrium 17	Aortic
8	64 M	Aortic stenosis; angina; obstructive airway disease	III angina	Aortic gradient 300 Cardiac index 1.8 Left atrium 30 Right ventricle 87/12	Aortic
9	65 F	Mitral insufficiency; atrial septal defect	III	None	Mitral
10	60 F	Mitral stenosis; mitral insufficiency; commissurotomy 1957; pulmonary emboli	IV	Cardiac index 2.7 Left atrium 27	Mitral

TABLE 1.—*Additional Clinical Data on Ten Cases*

Case No.	Bypass Time (minutes)	Postoperative Complications	Hospital Days	Follow-Up Data	Follow-Up Period (months)
1	103	Pulmonary embolus and myocardial infarction	32	Alive; Class I-II	14
2	182	Oliguria for two days	17	Alive; resistant atrial fibrillation; Class II	10
3	138	None	16	Alive; cardioversion; Class I-II	....
4	61	Metabolic alkalosis; stress ulcer; staphylococcus bronchitis	36	Alive; Class I-II	7
5	140	Arrhythmias; stress ulcer	25	Alive; Class II; slight aortic insufficiency	5
6	105	Low serum potassium; ventricular tachycardia and fibrillation	13	Alive; Class I	3
7	116	Died in operating room; right coronary artery not perfused	0	Died	17
8	128	Hematoma left groin	21	Alive, Class II; cardioversion	16
9	110	Brief ventricular fibrillation	11	Alive, Class I	16
10	108	None	10	Alive; cardioversion	15

to 26 resulted in apparently satisfactory perfusion as judged by: (1) Rare and minimal base deficit (metabolic acidosis); (2) increased urinary output during and after perfusion; and (3) prompt recovery of consciousness.

Technique. Valve replacement was done with the standard operative technique. The Starr-Edwards ball-valve prosthesis was used for both mitral and aortic replacement with the exception of Case 4 (Table 1). This patient had active bacterial endocarditis, and a Magovern sutureless valve was used to avoid a cloth cuff. Every patient but one (Case 7) had constant coronary perfusion throughout the intracardiac procedure. When aortic cross-clamping was required, self-retaining cannulas[6] were inserted in both coronary ostia for uninterrupted perfusion through a separate pump at 150 to 250 ml per minute, except in Case 9 where intermittent coronary perfusion was obtained by interrupted aortic cross-clamping with hypothermic protection (24.5°C).

Critical Periods

The management of this marginally compensated group is necessarily more demanding and precise than that of younger and less critically ill patients. The critical periods are: (1) During restoration of cardiac function and resumption of circulatory load; and (2) the postoperative period of 24 to 72 hours when myocardial dysfunction, distorted hemodynamics, impaired pulmonary function, fluid shifts, renal dysfunction and altered electrolyte patterns are present.

Operative Management

The first danger period begins with completion of the intracardiac procedure, when the heart is filled and defibrillated. Meticulous prevention of air embolism and ventricular distension is necessary since these phenomena may occur so quickly that they pass unrecognized. Goldfarb and Bahnson[2] demonstrated in dogs that an air embolus of only 0.1 ml will significantly impair myocardial function. We routinely vent the high point of the ascending aorta with a special needle similar to that described by Groves and Effler[3] and are frequently astonished by the number of bubbles that appear after supposedly careful filling of the heart.

Ventricular distension, usually caused by an incompetent aortic valve or prosthesis, or by a jammed ball resisting ventricular outflow, can result in prolonged or fatal impairment of ventricular function. To prevent this, a large-bore decompression tube should be maintained in the left ventricle until the critical period is past.

At the end of by-pass, restoration of circulating volume (adequate to drive an impaired ventricle) can be safely accomplished only with the help of left atrial pressure measurements. Experience has abundantly demonstrated the inadequacy of empirical blood replacement, since the heart with a low output may be fatally overloaded during an attempt to restore normal systemic arterial pressure. Conversely, normal blood volume may be inadequate to provide the elevated left atrial pressures which are frequently necessary to achieve maximum left ventricular output.

A weakly contracting heart may have a dramatic inotropic response to the administration of calcium ion or isoproterenol. Although these drugs may be needed only temporarily, they may be indispensable in restoring adequate coronary (and central) circulation, and in preventing progressive acidosis and ischemic fibrillation. They must be kept in readiness during this phase of the procedure.

Postoperative Management

During the second critical period immediately following operation, other potential dangers must be recognized and avoided.

Deficient Cardiac Output. Hypovolemia and impaired myocardial function are both potential postoperative problems and have indistinguishable clinical manifestations. Myocardial performance is temporarily depressed in many patients undergoing valve replacement and the heart may be driven into irreversible failure when blood is given to raise the arterial pressure. On the other hand, excessive caution in the postoperative replacement of blood (particularly when blood loss is unrecognized or miscalculated) may result in fatal shock. To avoid this dilemma and to guide postoperative blood administration precisely, left atrial pressure is monitored through a fine polyvinyl catheter brought out through the skin, as suggested by Kirklin and Theye.[4]

Inotropic Drugs. When depressed myocardial performance results in a progressive circulatory deficit, survival depends upon augmenting performance by inotropic drugs, by reducing metabolic needs, or both. We use a constant infusion

of diluted isoproterenol for this purpose because it has no undesirable vasoconstrictor effects, but occasionally patients will respond more satisfactorily to epinephrine.[5] Efficacy of the inotropic drug is easily demonstrated by the rise in left atrial pressure, drop in systemic pressure, metabolic acidosis and oliguria that occur when the drug is temporarily discontinued during the low output period.

Ventilatory Insufficiency. Hypoxia has been emphasized as a postoperative hazard in open-heart operations, and the need for assisted ventilation during this period is obvious. We have learned, however, that the patient's color is an unreliable indication of hypoxia. Frequent analysis of blood gas can be performed on samples withdrawn from the intra-arterial cannula used to monitor pressure. In most patients some degree of atelectasis develops, with pulmonary shunting and arterial desaturation for several hours postoperatively even when the pleural spaces remain intact. During this time the patient may have adequate alveolar ventilation (normal arterial pCO_2) without clinical cyanosis, but with significant hypoxemia. He may become insidiously acidotic without a constant high oxygen atmosphere and assisted ventilation, and "unexplained" ventricular fibrillation may develop.

Ventricular Fibrillation. In two patients serious arrhythmias and ventricular fibrillation developed, requiring prompt electrical depolarization. In Case 6 frequent extrasystoles led to fibrillation three times and remained a hazard until hypokalemia (3.0 mEq per liter) was corrected. Normal preoperative serum potassium and the 5 mEq of potassium present in the priming solution were inadequate to prevent hypokalemia. The patient would have died if this had not been recognized. Postperfusion hypokalemia occurred frequently in our series and is discussed in recently published reports.[1]

Results

Nine patients survived and all were improved to at least Class II; none had serious complications or lasting residual defects (Table 1). No thromboembolic complications, or hemorrhagic complications from anticoagulation, have occurred so far.

The one patient who died in this series was a 65-year-old woman (Case 7) with severe aortic stenosis complicated by angina pectoris at rest. A stenotic but patent right coronary ostium was seen on the angiocardiogram. During operation the ostium was gently dilated but attempts to perfuse it were unsuccessful. After the valve was replaced the ventricles could not be defibrillated. At autopsy an arteriosclerotic plaque was found to be separated from its base and acting as a flap valve at the origin of the right coronary artery. In retrospect this patient might have been saved if a suitable onlay graft had been used to enlarge the right coronary ostium at the outset of the procedure.

Discussion

This experience demonstrates that the potential hazards of prolonged and complicated intracardiac procedures can be avoided even in elderly patients. The mortality rate of 10 per cent in this series is comparable with that of 16 per cent in 75 younger patients who had single and multiple valve replacements during the same period. Our more recent experience of 2 per cent mortality in 46 consecutive patients of all ages further supports a more liberal attitude toward operation in the older patients.

Recognition and management of the cardiopulmonary problems associated with these operations are dependent upon attention to important details, particularly in aged patients.

Micro-air embolism is common and difficult to avoid; bubbles sometimes emerge from the vent for as long as ten minutes after restoration of ventricular output. When unexpected ventricular fibrillation or depressed output occurs during this period, air is often seen in a branch of the right coronary artery. The frequent occurrence of depressed myocardial performance following valve replacement is probably related to this insidious complication.

Monitoring of left atrial pressure has proved to be the only reliable guide for fluid (blood) replacement and for determining the need for inotropic drugs. We have treated borderline patients in whom adequate perfusion could be achieved only by transfusion to a left ventricular filling pressure of 25 cm of water. However, the further addition of as little as 100 ml of blood may produce a sharp rise in atrial pressure and pulmonary edema.

Adequacy of perfusion is poorly reflected by peak systolic blood pressure which is maintained by reflex vasoconstriction. Mean arterial pressure

monitored from an indwelling cannula is a more significant measurement but still does not correlate well with cardiac output. We firmly believe that peripheral vasoconstrictor drugs are contraindicated in the long-term management of cardiogenic shock because they cause metabolic acidosis. A clear sensorium, adequate urinary output, warm skin and palpable peripheral pulses reflect satisfactory circulation regardless of blood pressure. Absence of these signs should cause concern even if blood pressure is normal or elevated.

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